Table. Evolution of acute neuroschistosomiasis in 12-year-old boy after treatment

Parameter	Day 0	Day 45	Month 10
Neurologic symptoms	Present	Present but diminished	Absent
Eosinophil count (per mm^3)	3,080	1,030	370
Schistosoma ELISA	Weakly positive	Weakly positive	Negative
Schistosoma indirect hemagglutination assay (antibody titer)	Negative	640	80
Urine microscopic analysis	Normal	Not available	Normal
Urine concentration test for schistosomal eggs	Not available	Not available	Negative
Feces concentration test for schistosomal eggs	Not available	Not available	Negative

tially infected water. Furthermore, hypereosinophilia is an early warning sign, as seroconversion and egg excretion may be slower to evolve. Both elements provide sufficient circumstantial evidence to strongly suspect the diagnosis (*2*). In this case, the full-blown Katayama syndrome contributed to the evidence.

Praziquantel only kills adult worms and does not inactivate schistosomules, nor the miracidium inside the eggs, which will continue to elicit a damaging immunologic response for some time. Early antischistosomal treatment might, in fact, worsen symptoms (*7*). Because schistosomules may require up to 8 weeks to mature, early postexposure treatment with praziquantel cannot be used to forestall disease after primary infection. Furthermore, Katayama syndrome may occur as early as 3 weeks after exposure. On the other hand, withholding praziquantel until larvae have matured (8 weeks after exposure) would not prevent Katayama syndrome in many cases (*7*). Acute symptoms, including early neuroschistosomiasis, may therefore still develop during this 5-week window after exposure, despite early praziquantel administration.

Artemether has shown promising activity against schistosomules (*8*). Repeated administration throughout the transmission season has prevented Katayama syndrome in *S. japonicum* infection (*9*). Its use, singly or in combination with praziquantel, should be investigated as true postexposure prophylaxis for primary schistosomal infection in nonimmune travelers (*10*).

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Murine Typhus from Vietnam, Imported into Japan

To the Editor: In Vietnam, many febrile diseases such as malaria, dengue fever, Japanese encephalitis, scrub typhus, and more recently, severe acute respiratory syndrome (SARS) and avian influenza have been reported. Murine typhus cases were also reported during and before the 1960s but not thereafter (*1–5*).

On May 3, 2003, a 54-year-old male resident of Tokushima, Japan, had onset of fever in the suburban town of Cu Chi, ≈60 km northwest of Ho Chi Minh City, Vietnam. Exanthema appeared on his trunk and limbs on May 7. He returned to Japan on May 9 and was admitted to

Tokushima University Hospital on May 10. His body temperature was 39.0°C, and serum, C-reactive protein level was high (17.06 mg/dL) on admission (day 8 of illness). Unfortunately, the blood sample taken on that day was discarded. We then collected blood on days 10, 11, 12, 14, 17, and 24 of illness for diagnosis. Minocycline was administered on day 8 and resulted in a gradual decrease in fever and rash. Weil-Felix tests on day 12 showed the serum to be positive for *Proteus vulgaris* OX19 (titer 160); tests for *P. vulgaris* OX2 and OXK were negative (titer of 10 for both). We examined blood samples for possible diseases such as malaria, dengue fever, SARS, and rickettsioses. Giemsa-stained peripheral blood samples obtained on day 11 showed no malarial parasites. Results of immunoglobulin M (IgM)-capture ELISA of serum on days 10, 11, and 17 of illness were negative for dengue antibodies. Reverse transcription (RT)–PCR of the serum on day 11 was also negative. RT-PCRs of a pharyngeal swab and urine collected on day 11 were both negative for the SARS coronavirus. These specimens were also injected into Vero cells, and no cytopathic effects were generated. RT-PCR of these cultures was also negative for SARS coronavirus. Moreover, SARS antibodies were not found in serum samples on days 11 and 14 of illness. Serum was also tested for *Orientia tsutsugamushi* and *Coxiella burnttii* on day 12 to exclude scrub typhus and Q fever as diagnoses.

Indirect immunofluorescence tests for etiologic agents of spotted fever, murine typhus, and epidemic typhus were then performed with serum samples collected on days 10, 14, and 24. We used *Rickettsia typhi* and *R. prowazekii* as typhus group (TG) rickettsial antigens and *R. japonica* and *R. conorii* as spotted fever group (SFG) rickettsiae. IgM antibody was detected for these antigens, indicating that the disease was a primary infection of rickettsiae (Table). When TG and SFG rickettsioses were compared, TG rickettsiae represented markedly higher elevated titers than SFG rickettsiae, which excluded a diagnosis of SFG rickettsiosis. PCR for the TG rickettsial genome in the convalescent-phase serum on day 10 was negative.

To demonstrate more detailed antigenic reactivity, Western immunoblotting was performed with serum on day 14 (*6*). The serum reacted similarly to the ladderlike lipopolysaccharide (LPS) of *R. typhi* and *R. prowazekii*. As expected from the group-specific nature of rickettsial LPS, no reaction was demonstrated to LPS of SFG rickettsiae, *R. japonica* and *R. conorii*, although weak reactivity, mainly to the major outer member protein of SFG rickettiae, rOmpB, and molecules of smaller sizes was shown (*6*,*7*). As described previously, rOmpB has cross-reactive antigenicity between TG and SFG rickettsiae (*6*). Compared with the trace reaction to rOmpB of SFG rickettsiae, an extremely high level of reaction was demonstrated to rOmpB of TG rickettsiae. These results confirmed the disease to be a TG rickettsiosis.

To elucidate whether the disease was murine typhus or epidemic typhus, we conducted cross-absorption tests as described previously (*8*,*9*). Serum absorbed by *R. typhi* showed complete absorption, demonstrating no reaction to *R. typhi* or *R. prowazekii* (Table). However, the serum absorbed by *R. prowazekii* resulted in incomplete absorption, demonstrating no reactivity to *R. prowazekii* but some reactivity to *R. typhi*, which was left unabsorbed. Western immunoblotting with the serum absorbed by *R. prowazekii* showed reactivity only to the rOmpB of *R. typhi* but not to that of *R. prowazekii*. These results confirmed the diagnosis of murine typhus.

This is the first serodiagnosis of murine typhus in Vietnam since the 1960s (*1*–*5*). Since rats inhabit the area where the patient acquired the illness, murine typhus seems to have

Table. IFA titers of the patient sera and the cross-absorption test*

Day of illness	Immunoglobulin class	Antigen for absorption	Antigen for IFA titration			
			TG rickettsiae		SFG rickettsiae	
			R. typhi†	*R. prowazekii*‡	*R. japonica*§	*R. conorii*¶
10	IgG	(–)	320	320	<20	20
	IgM	(–)	160	40	20	20
14	IgG	(–)	1,280	640	<20	40
		R. typhi	<20	<20	<20	<20
		R. prowazekii	160	0	<20	<20
	IgM	(–)	640	320	80	80
		R. typhi	<20	<20	<20	<20
		R. prowazekii	160	<20	<20	<20
24	IgG	(–)	640	640	<20	40
	IgM	(–)	640	320	80	80

*IFA, indirect immunofluorescence assay; IgG, immunoglobulin G; TG, typhus group; SFG, spotted fever group.
†Strain Wilmington.
‡Strain Breinl.
§Strain YH.
¶Strain Malish 7.

occurred sporadically or endemically but to have been undiagnosed since the 1960s, maybe because it was thought to have been eradicated and thus widely forgotten. This case was the first imported into Japan since the 1940s, when many Japanese soldiers and residents who returned from abroad had the disease.

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Epidemic Risk after Disasters

To the Editor: We conduct communicable disease risk assessments after humanitarian emergencies, including natural disasters, and would like to clarify the findings of Floret et al. (*1*) regarding the risk for epidemics in certain disaster settings. Natural disasters that do not result in population displacement, regardless of type of disaster, are rarely associated with increased risk for epidemics. However, large-scale population displacement, with consequent overcrowding in temporary settlements and disruption of water supply and sanitation, are indeed associated with increased risks for communicable disease transmission. This distinction is well documented (*2–4*). Increased communicable disease incidence after flooding and cyclones has been particularly well described (*5*,*6*). In addition, after a disaster of any type, epidemics may go undetected because of poor surveillance or because baseline surveillance data for diseases (such as dengue fever or malaria) are unavailable.

Although we agree with the authors that media reports are often exaggerated and that the risk for epidemics after certain types of natural disasters (e.g., volcanic eruption) is low, we believe the findings are somewhat misleading. Postdisaster communicable disease incidence is related more closely to the characteristics of the displaced population (size, health status, living conditions) than to the precipitating event.

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